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Porcine Intestinal Adenomatosis: A Clinical Review

by Craig Rowles*
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INTRODUCTION

Porcine Intestinal Adenomatosis (PIA) is a disease affecting the alimentary tract of pigs.[1] Previous literature has described several syndromes relating to PIA, including Regional Ileitis[2-4] (RI), Necrotic Enteritis[5-7] (NE), and Proliferative Hemorrhagic Enteropathy[8-10] (PHE). However, it wasn't until recently that morphologic evidence was found, linking all four syndromes with a single etiologic agent.[11-12]

A clinical review of PIA, including incidence and prevalence, etiology, lesions, diagnosis, treatment and control, follows.

INCIDENCE AND PREVALANCE

PIA is reported to be worldwide in distribution. Cases have been documented in Australia, Canada, Denmark, Finland, India, Sweden, the United Kingdom, and the United States.[13] Two substantial slaughterhouse studies have been conducted to determine the percentage of swine showing lesions at slaughter.[2,14] Each indicated a low but appreciable level of lesions, .25% and < 1% respectively.

No literature exists relating the number or percentage of herds infected with PIA. However, it is generally agreed that this disease does pose a significant economic loss for swine producers.

Any age group may show clinical signs. However, it appears that at two different times a pig may be more likely to show clinical signs. During the post weaning period, pigs seem to show clinical signs relating to PIA, RI, and NE.[15] PHE, on the other hand, seems to strike 6 mo. −1 yr. old gilts and boars as they enter the breeding herd.

ETIOLOGY

Early observations of the disease were often hampered by other concurrent infections. Hence, an etiologic agent was difficult to identify. In 1972, however, a minimal disease herd broke with several cases of PIA.[8] Through the use of special culturing techniques[3] and immunofluorescence,[8] *Campylobacter sputorum* var *mucosalis* was identified. To date, this organism has not satisfied Koch's postulates. Yet, it is universally agreed that the disease would not occur without this vibrioid organism.[8,18]

Campylobacter sputorum var *mucosalis* is a gram negative, short, irregularly curved rod, 0.25 um wide and 0.95-2.8 um long. Sea gull, spiral and comma forms occur. Coccal forms also occur but are less prominent.[13]

Colonies grow on many solid nutrient media with 5-7% added blood. Microaerophilic conditions are necessary for growth. Colonies are 1.5-2.0 um in diameter after 48 hrs. and can be differentiated from other vibriones by the production of a yellow pigment and by tube aggulutination tests.[12,13]

Because this is an intracellular organism affecting the intestinal crypt cells, special culturing techniques must be used to isolate the organism from intestinal samples.[12] Most practitioners are not equipped to use this method of diagnosis.

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Another method used for identification includes the use of special staining of histologic sections, such as Young's modification of the Warthin-Starry stain.[19] This shows the organism in the intestinal crypt cells.

Immunofluorescence, using hyperimmune antivibrio serum, has been used sparingly as a method of identification in histologic sections.[12] However, the technique has not been perfected to the point of commercial use by diagnostic labs.

CLINICAL SIGNS

The clinical signs of PIA, RI, and NE are variable. Anything from decreased rate of gain with slight anorexia to severe wasting, complete anorexia and death may be seen.[13] Diarrhea is a common but not constant feature.[13]

Clinical signs of PHE are slightly different. One sees anorexia, depression and a severe melena marked by dark tarry feces.[16,17] In peracute cases, an animal may be found dead, due to severe hemorrhagic anemia.

Recovery is prolonged, taking 4-6 weeks. During this time the animals will be stunted, causing severe economic loss. However, when recovery does occur, the animals most frequently return to normal feed consumption and average daily gain.[13]

Morbidity/mortality rates have been documented in a single herd showing the PHE syndrome. These swine showed 12% morbidity and 6% mortality.[16]

LESIONS

The lesions associated with PIA vary with the particular syndrome involved. The following discussion will deal with the gross and histologic lesions of each syndrome.

Porcine Intestinal Adenomatosis is usually localized to the terminal portion of the ileum, but can be found in the cecum and upper spiral colon. The mucosa proliferates and is thrown into deep longitudinal or transverse folds. On the serosal surface this proliferation often assumes a "reticulated" pattern. Very little inflammation will be seen.[13]

Histologically, the epithelial proliferation is also seen. Large branching glands lined by immature columnar epithelial cells are seen. Many of these cells show mitotic figures, resembling a neoplastic lesion of man. Goblet cells are decreased in number and on rare occasions a few inflammatory cells are seen.[13]

Necrotic Enteritis is a more severe form of the disease. Grossly, the mucosa is covered by a yellow, necrotic membrane.[13] Ingesta is often seen attached to this membrane and deep fissures are evident.[11]

Histologically, coagulative necrosis of the mucosa is the most prominent feature. In many cases this necrosis is so severe, the original architecture is difficult to discern.[11]

Regional Ileitis is identified by the thickened appearance of the muscular coats of the terminal ileum, hence, the nickname "garden hose gut."[11]

Histologically, the thickening is due to hypertrophy of the muscular coats. In addition, the submucosa shows wide areas of granulation tissue. The mucosa is covered by necrotic debris.[11]

In Proliferative Hemorrhagic Enteropathy the ileum will be slightly dilated. The serosal surface shows the same reticulated pattern. Intralumenally, a large blood clot will be seen along the distal small intestinal tract, mixed with ingesta. The cecum and colon will show evidence of dark tarry feces.[8]

Histologically, the lumen shows variable amounts of blood and fibrin. The mucosa is thickened and the normal villous architecture is lost. The lamina propria is congested and edematous. Capillary beds will be disrupted but there will be no evidence of specific blood vessel wall damage.[8]

DIAGNOSIS

No single laboratory test exists to detect PIA in the live animal.[13] Therefore, diagnosis must be based on history, clinical signs, and complete necropsy of selected animals.

Confirmation of the diagnosis can be made by submitting affected portions of ileum for culture and histologic exam.

Differential diagnosis could include many enteric diseases of swine. PIA, NE, and RI can be mistaken for salmonellosis, or coccidial infections. The diarrhea and diphtheritic membrane formation characteristic of salmonella infections are similar to those clinical signs and lesions associated with PIA. However, the proliferative lesions are not observed. Coccidiosis also produces a necrotic enteritis syndrome. Laboratory diagnosis, using smears and histology, will rule this out.

Several hemorrhagic-like syndromes may mimic PHE, particularly gastric ulcers and

torsions of the mesentery.[20] Careful necropsy reveals congestion and thin walled, gas filled small intestines associated with torsions. No black tarry feces will be seen. Swine with gastric ulcers will have dark tarry feces throughout the length of the intestinal tract. Close observation will reveal the ulcer near the esophageal portion of the stomach. PHE will show mild proliferation of the mucosa as well as a characteristic blood clot in the ileum.[20]

TREATMENT AND CONTROL

Treatment of PIA is based on a whole herd antimicrobial therapy. Many antimicrobial agents have been claimed to be effective in the treatment of PIA. However, the following treatment regimes are generally agreed as being the most efficacious: (a) One regime calls for use of neomycin orally at the rate of 5 mg./lb. If used in the drinking water the rate is 1 gm./gallon; (b) Another antibiotic used is lincomycin in the feed at the rate of 100 gm./ton; (c) Lincomycin/spectinomycin in the drinking water has been used at the rate of .3 gm. lincomycin and .6 gm. spectinomycin/gallon; (d) Sulfonamides, in particular sulfathiazole, at the rate of 1 lb./120 gal. have also been used; (e) Finally, sodium arsenilate at the rate of 136 mg./gallon for 5 days has also been used. Levels of up to 600 mg./gallon can be tolerated for 5-7 days without problems of toxicity.[21]

In addition, every effort should be made to ascertain that new seed stock entering the breeding herd are not carriers. Isolate the animals for thirty days. Remove all antimicrobial agents from the feed. Next, provide across the fence exposure to suitable animals from your own herd. Finally, at the end of the thirty days, breed the animals. If the animals are carriers, the stress of this period should stimulate clinical signs. If any of the animals show clinical signs, all should be culled.

Of course, one cannot overemphasize the importance of good management techniques. Keeping a closed herd, minimizing traffic through the herd, and maintaining good sanitation and nutrition are important for prevention of PIA, as well as other diseases.

CONCLUSIONS

PIA is a disease that causes significant economic losses to swine producers. The role of *Campylobacter* in the disease is confirmed but further work needs to be done to determine what other factors are involved. Diagnosis, based on necropsy, culture, and histopathology, is a viable means of confirmation. However, the development of a fluorescent antibody test would greatly facilitate this diagnosis. Finally, until more is learned about this disease, work must be done to keep herds free of it by maintaining good management practices.

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